Signature: Med Sci Monit, 2003; 9(9): CR392-395
PMID: 12960928

Received: 2002.10.03
Accepted: 2003.07.14
Published: 2003.09.08

Gastric myoelectric activity in patients with closed head brain injury

Authors' Contribution:
A Study Design
B Data Collection
C Statistical Analysis
D Data Interpretation
E Manuscript Preparation
F Literature Search
G Funds Collection

Piotr J. Thor[1], Igor Gościński[2], Władysława Kolasińska-Kloch[3], Dorota Mądroszkiewicz[4], Ewa Mądroszkiewicz[2], Agata Furgała[1]

[1] Department of Pathophysiology, Collegium Medicum, Jagiellonian University, Cracow, Poland
[2] Department of Neurotraumatology, Collegium Medicum, Jagiellonian University, Cracow, Poland
[3] 2nd Clinic of Cardiology the Institute of Cardiology, Collegium Medicum, Jagiellonian University, Cracow, Poland
[4] Department of Gastroenterology, Collegium Medicum, Jagiellonian University, Cracow, Poland

Source of support: This study was supported by grant no. 501/P/79/L from the State Committee for Scientific Research (KBN), Warsaw, Poland.

Summary

Background: Traumatic brain injury (TBI) constitutes a major public health problem. Most of the acute disturbances of autonomic nervous system activity seen in clinical practice resulting from head injury are due to increased intracranial pressure (ICP), which precipitates a complex set of changes causing lack of food tolerance. The aim of our study was to evaluate the effects of TBI on gastric myoelectric activity in coma patients.

Material/Methods: 24 patients (23 male, 1 female; mean age 43.6±7 yr.) hospitalized for TBI in the Department of Neurotraumatology were included in the study. Gastric myoelectric activity was recorded using the Synectics system (Sweden) on the 1st and 2nd day after injury. The control group consisted of healthy volunteers matched for age and gender.

Results: In the control group, electrogastrography (EGG) showed the percentage of time with bradygastria, normogastria and tachygastria to be 9.7±6.4, 88.3±7.6 and 2.0±3.5 respectively. The amplitude of the signal was 181±11.5 [μV^2]. In TBI patients, the percentage of time with bradygastria and tachygastria increased to 46.5±21.8 ($p=0.03$) and 5.5±11.8 ($p=0.001$) respectively, while the duration of normogastria decreased to 47.2±20.4 ($p=0.001$). Highly significant changes were observed in signal amplitude, which increased to 766±550 [μV^2] ($p=0.0007$). There was positive correlation between GCS and normogastria ($r=0.66$, $p<0.001$) and negative correlation between GCS and bradygastria ($r=-0.77$, $p<0.001$).

Conclusions: In patients with brain trauma and coma, the functional brain-gut link is altered, causing gastric dysrrhythmias and intolerance to feeding. Prokinetics during enteral feeding are recommended.

key words: traumatic brain injury • increased intracranial pressure • gastric myoelectric activity

Full-text PDF: http://www.MedSciMonit.com/pub/vol_9/no_9/3180.pdf

Word count: 1424
Tables: 1
Figures: 4
References: 20

Author's address: Professor Piotr J. Thor, Department of Pathophysiology, Medical College, Jagiellonian University, ul. Czysta 18, 31-121 Cracow, Poland, email: mfthor@cyf-kr.edu.pl

BACKGROUND

Gastric motility and emptying is governed not only by peripheral myogenic activity (pacesetter activity), but also remains under the control of autonomic innervation located centrally in the autonomic brain centers. Traumatic brain injuries (TBIs) vary considerably in their etiology, pathophysiology and clinical presentation. For classification purposes, TBIs are usually divided into focal and diffuse. Focal brain injuries include contusions, brain lacerations, and hemorrhage leading to the formation of hematoma in the extradural subarachnoid, subdural or intracerebral compartments [1]. Diffuse brain injuries involve cerebral contusion, edema, diffuse axonal injury, and prolonged posttraumatic coma. Trauma of the brain involves neural or vascular elements, which results in axonal damage of the Ranvier nodes, ischemia, cerebral edema and increased intracranial pressure (ICP) [2,3]. However, the effects of TBI on gastric motility and the lack of food tolerance is described in the literature in conflicting terms. For example, Livingston [4] found that increased ICP stimulated gastric contractility, but the opposite effect was observed by Garrick [5].

It is well known that gastric myoelectrical activity is highly correlated with gastric motility [6,7]. The non-invasive recording of gastric myoelectric activity using skin electrodes has become lately a very popular research and clinical tool in humans. The summary of recorded gastric myoelectrical activity is called an electrogastrogram (EGG). The frequency of gastric contractions is controlled by the gastric slow wave, and the amplitude of gastric contractions is associated with higher cholinergic drive [7]. Central damage to the autonomic system caused by TBI, resulting in autonomic dysfunction, should be expected to affect gastric myoelectric activity.

The aim of our study was to evaluate the effects of traumatic brain injury on gastric myoelectric activity in comatose patients.

MATERIAL AND METHODS

We studied 24 patients, 23 male and 1 female (mean age 43.6±7yrs), in coma after a closed head injury, on the first or second day after trauma. Six of them had diffuse brain damage, six had extradural and six subdural hematoma, and six patients had intracerebral hematoma. Their clinical status on the Glasgow Coma Scale (GCS) was between 4 and 7 points. Fasted antral myoelectric activity was captured in all patients with standard cutaneous electrodes (Synectics, Sweden) and the data stored on the hard disc of a computer. The collected data were analyzed and presented graphically (software from Synectics Medical AB, Stockholm, Sweden). The patterns of gastric myoelectric activity were analyzed with regard to the percentage time of bradygastria (frequency 0.5–2.0 cycles per minute, cpm), normogastria (2–4 cpm), and tachygastria (4–10 cpm). The following parameters were also calculated:

- PDF – Period Dominant Frequency, the dominant frequency (highest peak) of the mean or average FFT (Fast Fourier Transform) line;
- PDP – Period Dominant Power, the power or amplitude of the PDF peak;
- DFIC – Dominant Frequency Instability Coefficient, also based on the dominant frequencies of the FFT lines that make up a given period. The DFIC indicates dominant frequency changes over the course of the period. It is computed by first calculating the mean and standard deviation of the individual dominant frequencies for the period in question. The standard deviation of the dominant frequencies is then divided by the mean of the dominant frequencies to give the DFIC, which is reported as a percentage;
- DPIC – Dominant Power Instability Coefficient, based on the powers of the dominant frequencies over the given period. The DPIC is a measure of how much the power of the dominant frequency changes over the course of the period.

24 healthy volunteers without neurological symptoms matched for age and gender served as a control group. Patients with obesity, hypertension, diabetes mellitus or valvular heart disease were excluded from the study. No subjects had a prior history of gastrointestinal tract diseases.

The research was performed between January 1999 and January 2001 in the Neurotraumatology Clinic at the Jagiellonian University College of Medicine. The project was approved by the Jagiellonian University's ethical review committee, and was performed in accordance with the Helsinki Declaration.

Statistical analysis

For statistical analysis we used Statistica software (Statsoft, USA). Data are presented as mean values (SD) A P value <0.05 was considered significant.

RESULTS

In the control group, the percentage of recorded time with bradygastria, normogastria and tachygastria was 9.7±6.4%, 88.3±7.6% and 2.0±3.5% respectively. The fasting amplitude of signal was 181±11.5 [μV^2]. In the TBI patients, the percentage of time in bradygastria and tachygastria increased to 46.5±21.8% ($p=0.03$) and 5.5±11.8% ($p=0.001$) respectively, at the cost of normogastria, which was reduced to 47.2±20.4% ($p=0.001$) (Figure 1). The amplitude of signal was higher in the TBI patients (766±55 [μV^2], $p=0.0007$) than in the control group (Figure 2). Amplitude increased about 200% in the control group after a meal, to 366±38.4 [μV^2]. The PDF, PDP, DPIC, DFIC values are presented in Table 1.

There was positive correlation between the Glasgow Coma Scale score and normal frequency timing (Spearman's rank correlation $r=0.66$; $p<0.001$) (Figure 3), and negative correlation between bradygastria timing and GCS ($r=-0.77$; $p<0.001$) (Figure 4).

DISCUSSION

In the functional division of the stomach the proximal and distal parts are differentiated. The distal stomach is

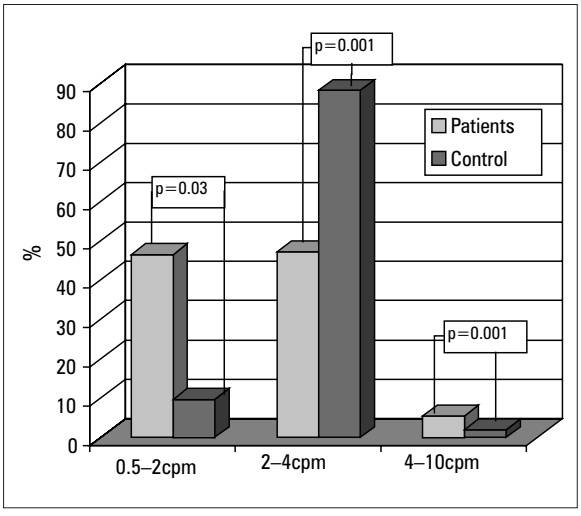

Figure 1. EGG changes in frequency timing in TBI patients and controls.

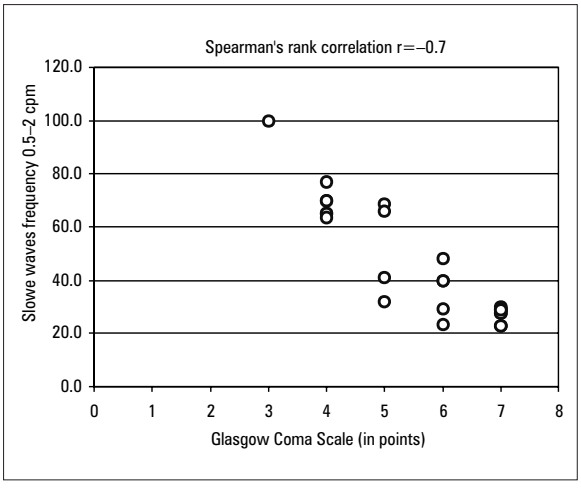

Figure 3. Correlation between Glasgow Coma Scale score and percentage of time recording slow wave frequency of 0.5–2 cpm.

Table 1. EGG parameters in TBI patients and controls.

Parameters	Controls	Patients	p*
PDF	2.7±0.8	2.07±1.29	0.02
PDP (μV2)	403±230	1711±420	0.002
DPIC (%)	34.2±18	61±35	0.0023
DFIC (%)	65±33	120±53	0.0006

Level of statistical significance: p<0.05

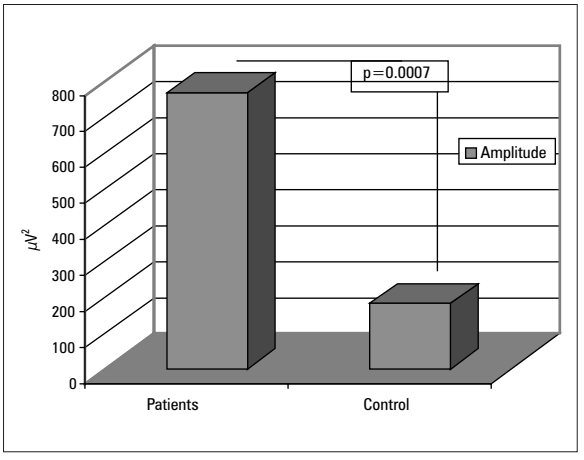

Figure 2. Signal amplitude recorded in TBI patients and controls.

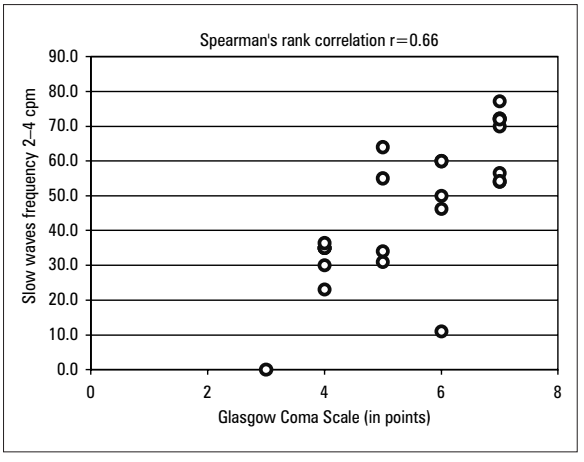

Figure 4. Correlation between Glasgow Coma Scale score and percentage of time recording slow waves frequency of 2–4 cpm.

governed by a basic electrical rhythm (slow waves) generated by a gastric pacesetter localized 2/3 of the way along the gastric curvature. The proximal part is responsible for the active reception of the meal and produces tonic contractions responsible for liquid gastric emptying. The distal part, by its peristaltic activity driven by the pacesetter-dependent rhythm (2–4 cycle per min, cpm), is responsible for gastric emptying of solids. Both parts are under well-balanced stimulatory /inhibitory control from the autonomic central nervous system (ACNS). Gastric myoelectric activity reflects gastric motility, recorded by measuring gastric slow wave frequency and the amplitude of the EGG signal [6]. From the theoretical point of view, gastric motility and emptying disturbances in TBI patients may be caused by the head injury itself (axon damage in the ACNS) or transmitted peripherally by nerves to the viscera or/and chemical mediators, which are released locally from nerve terminals in these patients [3,8,9]. Studies on animals have recently demonstrated that releases of excitatory amino acids play a major role in neuronal damage after brain injury and ischemia [10]. Trauma victims demonstrate severely decreased bowel sound, gastroparesis, and lack of cardiac response to tracheal stimulation and high gastric retention. These symptoms of peripheral autonomic dysfunction related to lesions of the dorsal vagus nucleus suggest vagal dysfunction [11]. Garrick found that peripheral cholinergic stimulation with betanechol reversed the inhibition of gastric and duodenal motility in response to increased ICP only for a short period of time. These short-lasting effects of betanechol confirmed mainly failure of the cholinergic system [5]. Kacker also claimed that intracranial hypertension is responsible for suppression of vagal activity

and inhibition of gastric emptying [12]. We found that gastric dysrrhythmias are related to GCS: a low GCS score is associated with a higher percentage of dysrrhythmias and vice versa, a higher GCS score correlates with normal frequency timing. This is important, because both types of dysrrhythmias of the gastric pacesetter cause delay in gastric emptying. The autonomic dysfunction induced by trauma could be the main reason for the observed gastroparesis [13]. Wood et al. also observed prolongation of gastric emptying for both phases of meal and antral hypomotility, with activity fronts appearing simultaneously in the proximal small intestine [14]. In our data the increase in dysrrhythmia time was associated with an increase in the amplitude of the slow waves, indicating stronger antral contractile activity, which contradicts Wood's observations in TBI patients. The underlying mechanisms of this observation are unclear [15,16]. Van Miert and De La Parra suggest that released endotoxins and liposaccharides after trauma are strong inhibitors of gastric emptying in rats [17]. It has been found that in patients with head injury the levels of interleukin-1 in the cerebrospinal fluid (CSF) are significantly elevated when compared to the level in lumbar CSF from patients undergoing routine myelograms [18]. Studies done by Tache [19] suggest that intracisternal corticotropin–releasing factor probably acts centrally to delay gastric emptying through brain sites that have not yet been determined. Other investigators have found a feedback loop between cytokines and CRF levels [17,20]. The issue of the mechanisms of the gastric motility changes in TBI patients remains open. It is probably the result of both direct neuronal damage and functional changes resulting from the release of inflammatory mediators, which in concert delay food transit in the gastrointestinal tract. Thus the use of prokinetics in these patients seems reasonable.

CONCLUSIONS

Our results provide evidence that in patients with brain trauma and coma, the functional brain-gut link is altered, causing gastric dysrrhythmias and intolerance to feeding. This suggests the use of prokinetics in unconscious patients on enteral feeding. The mechanisms of these disturbances remain unclear. The hypotheses that they are the result of neuronal damage or of functional changes due to release of inflammatory mediators require further studies.

REFERENCES:

1. MacQueen BD: Imagining the brain. Med Sci Monit, 2001; 7(4): 716-724
2. Clifton GL, Robertson CS, Grossman RG et al: The metabolic response to severe head injury. J Neurosurg, 1984; 60: 687-696
3. Valadka AB, Gopinath SP, Robertson CS: Midline shift after severe head injury; pathophysiologic implication. J Trauma 2000; 49 (1): 1-8
4. Livingstone EH, Passaro EP, Garrick T: Elevated intracranial pressure stimulates gastric contractility in the rat. J Surg Res, 1999; 50(2): 106-110
5. Garrick T, Mulvihill S, Buack M et al: Intracerebroventricular pressure inhibits gastric antral and duodenal contractility but not acid secretion in conscious rabbits. Gastroenterology, 1988; 95: 26-31
6. Chen J, McCallum RW, Richards R: Frequency components of the electrogastrogram and their correlations with gastrointestinal contractions in humans. Med Biol Eng Comput, 1993; 31: 60–67
7. Chen JD, Lin Z, Pan J, McCallum RW: Abnormal gastric myoelectrical activity and delayed gastric emptying in patients with symptoms suggestive of gastroparesis. Dig Dis Sci, 1996; 41(8): 1538-1545
8. Ott l, Young B, Ohillips R et al: Altered gastric emptying in the head injured patient: relationship to feeding intolerance. J Neurosurg, 1991; 74: 738-742
9. Armstead W: NOC/oFQ activates PKC and generates superoxide to impair hypotensive cerebrovasodilation after hypoxia/ischemia. Med Sci Monit, 2002; 8(4): 31-38
10. Bullock R, Zauner A, Woodward JJ et al: Factors affecting excitatory amino acid release following severe human head injury. J Neurosurg, 1998; 89(4): 507-518
11. Haig AJ, Ho KC, Ludwig G: Clinical, physiologic, and pathologic evidence for vagus dysfunction in a case of traumatic brain injury. J Trauma Injury Inf Critical Care, 1996; 40(3): 441–444
12. Kacker V, Mehta VS, Gupta YK: Acute intracranial hypertension-induced inhibition of gastric emptying: evaluation in conscious rats. Eur J Pharmacol, 1999; 369: 65-72
13. Altmayer T, O'Dell MW, Jones M et al: Cisapride as a treatment for gastroparesis in traumatic brain injury. Arch. Physical Med Rehab, 1996; 77(10): 1093-1094
14. McArthur CJ, Gin T, McLin IM et al: Gastric emptying after brain injury: effects of choice of sedation and intracranial pressure. Intensive Care Medicine, 1995; 21(7): 573-576
15. Norton JA, Ott LG, McClain C et al: Intolerance to enteral feeding in brain-injured patients. J Neurosurg, 1988; 68: 62-66
16. Wood JR, Camilleri M, Low PA, Malagelada JR: Brainstem tumor presenting as an upper gut motility disorder. Gastroenterology, 1985; 89: 1411-1414
17. Nompleggi D, Teo TC, Blackburn GL: Human recombinant interleukin-1 decreases gastric emptying in the rat. Gastroenterology, 1988; 94: 326 (Abstract)
18. Van Miert ASJ, De La Parra: Inhibition of gastric emptying by endotoxin in conscious rats and modification of this response by drugs affecting the autonomic nervous system. Arch Int Pharmacodyn Ther, 1970; 184: 27-33
19. Tache Y, Maeda-Hagiwara M, Turkelson CM: Central nervous system action of corticotropin-releasing factor inhibits gastric emptying in rats. Am J Physiol, 1987; 253: 241-245
20. Young AB, Ott LG, Beard D et al: The acute phase response of the brain-injured patient. J Neurosurg, 1988; 69: 375-380